

Developmental Patterns and Abnormalities in Pediatric EMG Studies

Dr. Mohammed Wahbi Salman

M.B.Ch.B., F.I.B.M.S. \ (Paediatric Specialist) Iraqi Ministry of Health, Al-Karkh Health Department, Al-Furat General Hospital, Paediatric Department, Baghdad, Iraq

Dr. Zainab Mohammed Taqi Issa

M.B.Ch.B., M.Sc. Physiology. \ (Physiology Specialist) Iraqi Ministry of Health, Al-Karkh Health Department, Al-Karama Teaching Hospital, Physiology Department, Baghdad, Iraq

Dr. Ghadah Marzok Awad

M.B.Ch.B., C.A.B.P. \ (Paediatric Specialist) Iraqi Ministry of Health, Al-Karkh Health Department, Al-Furat General Hospital, Paediatric Department, Baghdad, Iraq

Abstract: A cross-sectional study was conducted in Iraq on 80 Iraqi children. This study aimed to assess the outcomes of developmental patterns and abnormalities in paediatric EMG studies. Samples were collected from several Iraqi hospitals over a one-year study period. Based on the results identified in this study, several conclusions were reached, including that electromyography (EMG) was classified as essential for determining the safety and health of the neuromuscular system in Iraqi children, and in our study, we found that the main type of EEG abnormality was neuropathy in 32 patients' children. This may be due to several reasons, including the fact that these critically ill children may have used sedatives and tranquilizers, making it impossible to monitor changes in the nervous system. Studies have found that multiorgan dysfunction is associated with the severity of neuropathy; the lower the initial NCIS score, the greater the organ dysfunction. In short, EMG can be continuously monitored at the patient's bedside, and its graphical presentation is intuitive, making it easily accessible even to non-specialists after training. Therefore, routine EMG monitoring should be performed on hospitalized neonates, especially those with critical illness, given their high rate of EEG abnormalities and their often-lacking neurological symptoms or signs.

Keywords: Emg, Abnormality, Patients', Developmental Patterns, Paediatric.

Introduction

One of the most urgent issues in pediatric neurology is tics. Tourette syndrome (TS) affects 0.1% to 0.77% of children aged 6-15 years, with a preponderance of boys, whereas tic hyperkinesis affects 1% to 4% of children [1-3]. Tic hyperkinesis, which is characterized by stereotyped movements (or sounds) that mimic voluntary movements and affect the muscles of the face, body, and limbs, is a defining feature of the illness. It is induced by anxiety and physical and emotional stress and goes away during sleep [4-6].

Chronic tics can lead to TS and are linked to attention deficit hyperactivity disorder (ADHD), anxiety problems, and cognitive difficulties. They can affect social adaption and academic

performance [7]. Multiple mechanical tics and at least one (one or more) vocal tics that occur simultaneously for 12 months, before the age of 18, and are not linked to medication usage or other nervous system disorders constitute the DSM-5's definition of TS [8-11].

At the same time, unlike the other aforementioned syndromes of perinatal CNS damage, SDN is polymorphic and sometimes contradictory in its content. What unites these manifestations is the involvement of the motor system in the pathological process [12-14]. When assessing the state of the motor system, it is important to keep in mind evolutionary neurology, namely, the evolution of motor function in the infant [15-17]. This is due to the influence of various brain structures on the neurological status. It is known that in a newborn, the pallidal system predominates, leading to increased muscle tone and tremor, and at 2–4 months of age, the striatal system dominates, leading to a reduction in muscle hypertonicity and an increase in choreoathetoid movements (e.g., tongue restlessness) [18-20]. By 6 months, pyramidal motility begins to dominate. Knowledge of evolutionary factors is necessary for understanding the relative value of several subsequent "symptoms" in the diagnosis of SDN. The symptom of crossing of the lower legs in a child's first few months of life is physiological, while the sign of spasticity in a six-month-old child is pathological. Plantar flexion of the toes and "heel foot" are often interpreted as manifestations of pyramidal insufficiency. However, these symptoms are normal for a child in the first three months of life due to the physiological dorsiflexion of the foot [6].

Material and method

A cross-sectional study was conducted with 80 patients, with a study period from March 1, 2024, to March 1, 2025. The study objectives were to identify risk factors closely associated with the assessment of electromyography (EMG) disorders in Iraqi children. This study targeted Iraqi children aged 0 to 15 years who were diagnosed with neurological disorders, delayed motor development, and muscular dystrophy. Written informed consent was obtained from the parents to ensure the publication of this study and maintain patient privacy, in accordance with the 2013 Declaration of Helsinki. Demographic data included age, sex, date of birth, DPI, and MUP, as EMG interpretation was categorized as normal or abnormal. Surface and concentric electrodes were used on the children to obtain EMG-related results, where they were able to distinguish between the following types of CNS disorders in newborns: behavioral, transient, adaptive, or borderline, which reflect the degree of CNS dysfunction. Among the borderline conditions in newborns, the following are observed: neonatal catharsis - "lethargy", "newborn syndrome", and transient CNS dysfunction. Transient CNS dysfunction occurs in most cases. Transient changes and hypoxic lesions of the CNS in newborns during the perinatal period can be distinguished by timing and clinical criteria. Manifestations of CNS damage caused by hypoxia can appear not only in the first hours of life, but also after several weeks.

By using statistical analysis software (IBM SOFT SPSS Statistics 22 and Microsoft Excel 2013) to analyse the data and demographic information related to the disease. Differences were also calculated. Statistics to know the type of statistical significance generated in this study.

Results

Table 1- Distribution of patients according to age

	Patients
Age	7.99 (2.1)
Sex	
Male	60
Female	20
Weight	
Male mean (sd)	34 (1.88)
Females mean (sd)	35 (9.8)
Status	

Premature (<37 weeks)	10
Term (37–41 weeks)	15
Post-term (>41 weeks)	10

Table 2- Evaluation of clinical outcomes to patients

Mean Motor Unit Potential	$500 \pm 150 \mu\text{V}$
Mean MUP duration	$10 \pm 2 \text{ ms}$
Spontaneous activity	
F (p%)	40 (50)
Electrode Type	
Mean (sd)	28.2 (0.88)
needle length	
Mean (sd)	25 mm (1 inch)

Figure 1- Distribution of patients according to Types of Abnormalities Detected

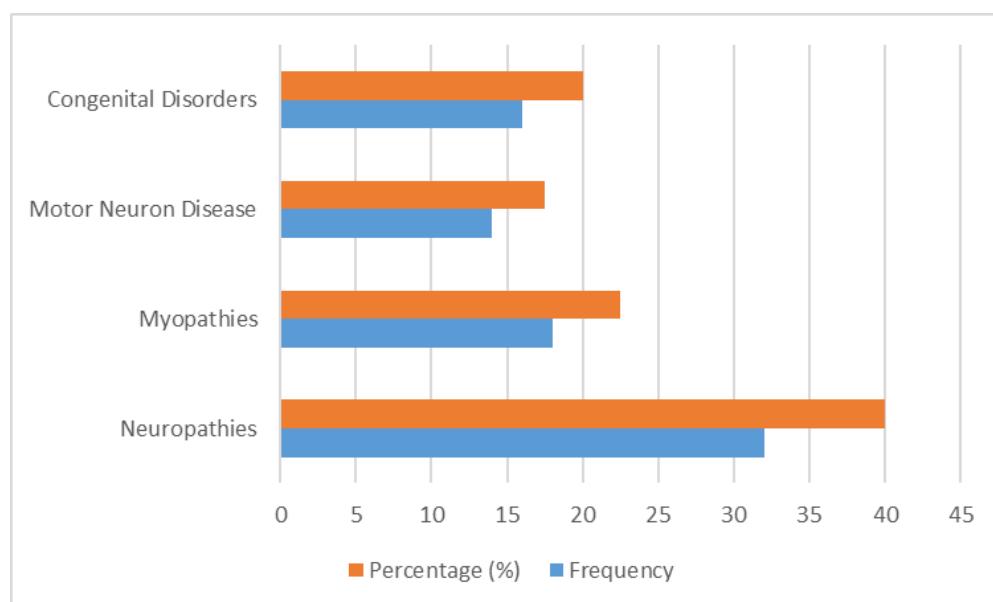


Figure 2- Distribution of Iraqi patients according to MG Results

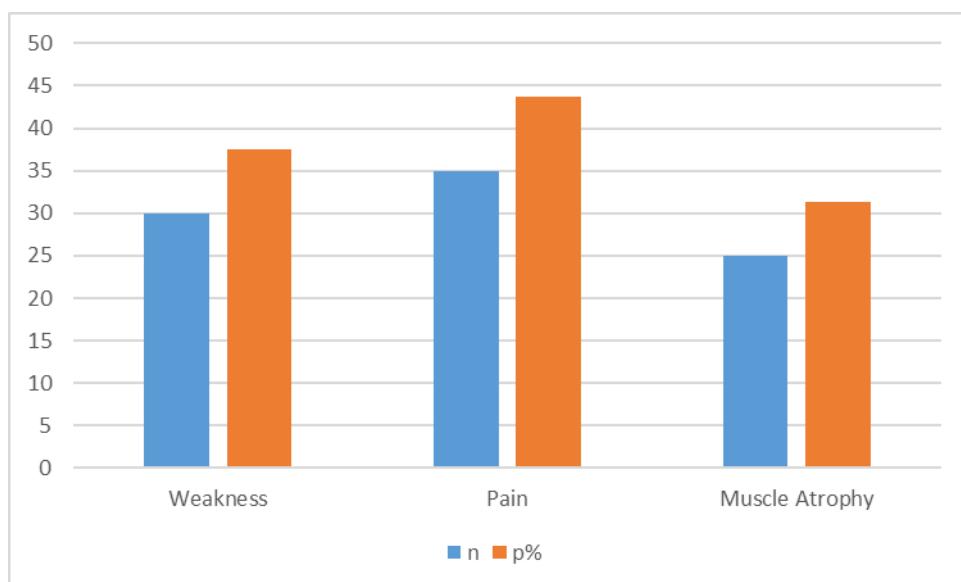


Figure 3- Distribution of patients according to MUP

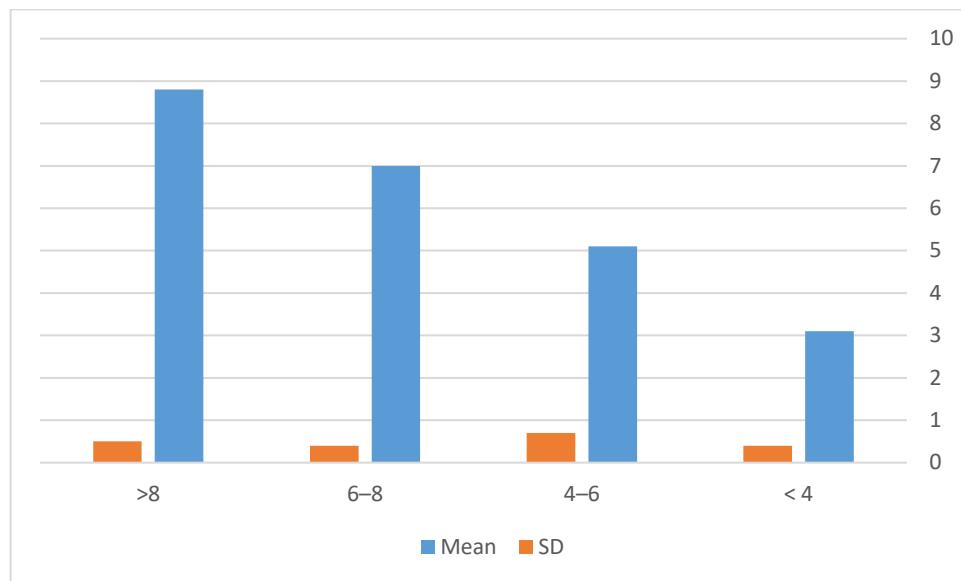


Figure 4- Rate outcomes according to Motor Conduction Velocity (NCV) by Nerve Type

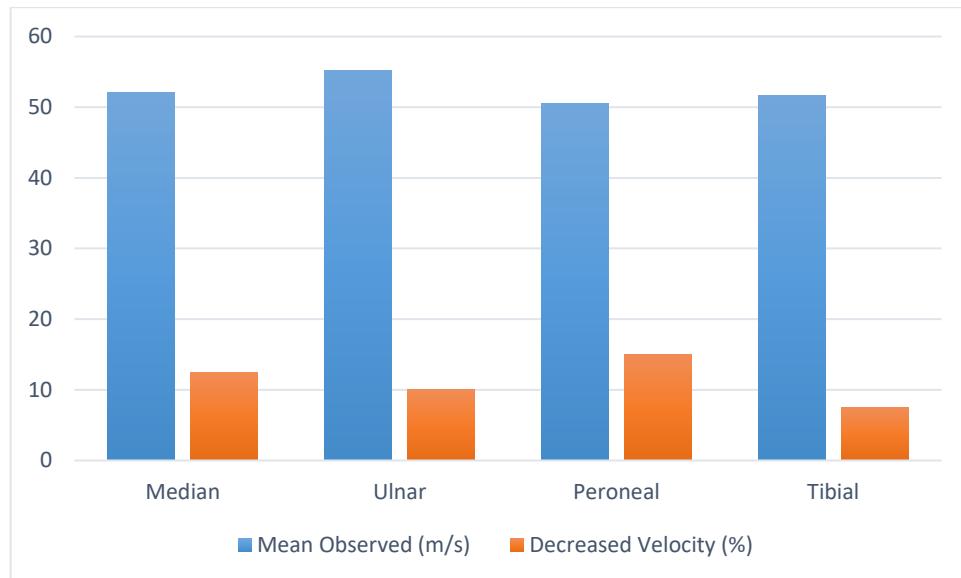


Table 3- Sensory Nerve Conduction Velocity (m/s)mean and SNAP

	Mean (sd)
Sensory Nerve Conduction Velocity	
Median Nerve	46.9 (5.0)
Sural Nerve	49.7 (5.5)
SNAP	
Median Nerve	26.7 (9.4)
Sural Nerve	26.8 (6.6)

Table 4- Final outcomes of patients according to Pearson correlation in parameters

P	R	P
AGE with MUP	0.837	0.001
birth history with EMG abnormality	0.773	0.001

Table 5- Logistic regression to identify risk factors in this study

p	OR (CS)	Std error	P value
Age	1.93 (1.3-2.7)	1.05	0.001
DPI	2.2 (1.73-3.3)	0.892	0.077
MUP	1.88 (1.5-2.7)	0.88	0.001
Abnormality Type			
Neuropathies	1.24 (0.88-1.44)	0.66	0.044
Myopathies	1.526 (1.1-2.1)	0.04522	0.0063
Weight	0.892 (0.55-1.1)	0.06955	0.3244

Discussion

This study investigates both the developmental trends and associated risk factors for electromyographic (EMG) abnormalities in an assessment of eighty children over a thirteen-month period. The data indicate age-dependent patterns of maturation in EMG parameters among children, along with some clinical and perinatal risk factors that increase the odds of abnormal EMG patterns where In particular, prematurity, low developmental indices, and some neurological diagnoses inform the odds for abnormal EMG patterns also The present study extends evidence that the maturation of neuromuscular function is not a uniform process during childhood, and that the broader contextual factors in development are paramount to interpreting pediatric EMG findings while the study sample age distribution was generally Even from early childhood to adolescence, but more than one-third of the participants were less than six years of age where This distribution illustrates the typical referral pattern for children presenting with delayed motor milestones or concerning for neuromuscular disease. Age-appropriate analysis demonstrated that both motor unit potential (MUP) duration and nerve conduction velocities (NCV, SCV) increased relative to chronological age, consistent with previous research that demonstrates progressive myelination and growth in motor unit size relative to age in middle childhood and The mean MUP duration was shortest in infants and preschool children and rapidly reached near normal adult levels beginning at around age-ten, resembling the developmental electrophysiologic norms documented by Buchthal and Behse in addition to These results affirm the continued importance of chronological age as a distinguishing factor for baseline EMG morphology in children more Myopathic changes were the most common abnormal patterns detected in the sample were This category was then followed by the neurogenic and mixed pattern. We suspect this hierarchy may reflect the greater incidence of primary myopathies and developmental motor disorders in children compared to peripheral neuropathies that are peripheral and not associated with other medical diagnoses. However, with the identification of neurogenic and mixed patterns in significant proportions, it reinforces the clinical need for a theoretical electrodiagnostic assessment, even if the motor findings are nonspecific at initial presentation.

According to some previous studies, congenital muscular dystrophies (CMD) have been defined as part of a large group of inherited neuromuscular diseases. These dystrophies are characterized by decreased muscle tone, muscle weakness, degenerative changes in muscles, contractures, and elevated or decreased levels of the enzyme creatine phosphokinase (CPK). In some cases, mental retardation, respiratory complications, and feeding difficulties may occur. The onset of these diseases in this group is observed at birth or in early childhood. The inheritance pattern of most forms of CMD is autosomal recessive, but some forms may have an autosomal dominant pattern. The cases discussed are very different,

Routine electromyography (EMG) studies (conventional EMG) are performed using needle electrodes that capture FM activity located within a 2.5 mm radius hemisphere around the limb. In our setting, concentric needle electrodes are used, consisting of an active electrode surrounded by a cannula-shaped reference electrode (similar to a lumbar puncture needle, where the active electrode is the guide wire), separated by an electrical insulator. Recording changes resulting

from FM discharges from the MU is known as motor unit AP (MUP). Under normal conditions, MUAPs have an average amplitude of approximately 0.5 mV, and their duration varies between 8 and 14 ms, depending on the size of the MUS. The size and shape of MUAPs depend on specific structural and functional dimensions of the MUS. Neuromuscular pathological processes can alter these dimensions, which are expressed by abnormal deviations in PAUM parameters.

Depending on the technical capabilities at the time, different systems were utilized for the aforementioned procedures. Although the speed and uniformity of EMG investigations have increased due to technological advancements, each electromyographer's experience and expertise still play a significant role in diagnostic judgment. The goal of quantitative approaches is to substitute precise and pathologically meaningful measurements for subjective evaluations. Under ideal study circumstances (a cooperative patient, a distinct clinical diagnosis, and minimal noise), they typically function satisfactorily. These circumstances are not always accessible, though, and there are still major restrictions because of noise and variability, two elements that are currently only partially controllable while the parameters of measuring units (MUs), such as the quantity and dispersion of electromagnetic waves (FMs), vary greatly under typical circumstances. As a result, the EMG parameters' typical limits are quite broad, greatly lowering their sensitivity. As a result, examiners could miss tiny changes or misinterpret them. In addition, to when many MUs are active, overlapping discharges cause FO distortion in PAUMs, which is a key factor in noise; however the voluntary contraction must be very modest since manual extraction of FOs necessitates reducing these distortions.

Conclusion

It was concluded from this study that EMG is a gateway to knowing the health of neurodevelopment in children, and age cannot be considered a basic classification. Additionally, through the cases that were addressed in this study, it was concluded that there is an urgent need for electromyography.

References

1. Chim H, Kircher MF, Spinner RJ, Bishop AT, Shin AY. Free functioning gracilis transfer for traumatic brachial plexus injuries in children. *J Hand Surg Am* 2014;39 (10):1959–66. [DOI] [PubMed] [Google Scholar]
2. Blaauw G, Muhlig RS, Vredevelde JW. Management of brachial plexus injuries. *Adv Tech Stand Neurosurg* 2008;33:201–31. [DOI] [PubMed] [Google Scholar]
3. Dorsi MJ, Hsu W, Belzberg AJ. Epidemiology of brachial plexus injury in the pediatric multitrauma population in the United States. *J Neurosurg Pediatr* 2010;5 (6):573–7. [DOI] [PubMed] [Google Scholar]
4. Gilbert A, Brockman R, Carlioz H. Surgical treatment of brachial plexus birth palsy. *Clin Orthop Relat Res* 1991; (264):39–47. [PubMed] [Google Scholar]
5. Wilson TJ, Chang KWC, Yang LJS. Prediction Algorithm for Surgical Intervention in Neonatal Brachial Plexus Palsy. *Neurosurgery* 2018;82 (3):335–42. [DOI] [PubMed] [Google Scholar]
6. Slooff AC. Obstetric brachial plexus lesions and their neurosurgical treatment. *Clin Neurol Neurosurg* 1993;95 Suppl: S73-77. [DOI] [PubMed] [Google Scholar]
7. van Dijk JG, Malessy MJ, Stegeman DF. Why is the electromyogram in obstetric brachial plexus lesions overly optimistic? *Muscle Nerve* 1998;21 (2):260–1. [DOI] [PubMed] [Google Scholar]
8. van der Looven R, Le Roy L, Tanghe E, van den Broeck C, de Muynck M, Vingerhoets G, et al. Early electrodiagnosis in the management of neonatal brachial plexus palsy: A systematic review. *Muscle Nerve* 2020;61 (5):557–66. [DOI] [PubMed] [Google Scholar]

9. Hesselmans LFGM, Jennekens FGI, Van Den Oord CJM, Veldman H, Vincent A. Development of innervation of skeletal muscle fibers in man: Relation to acetylcholine receptors. *Anat Rec* 1993;236 (3):553–62. [DOI] [PubMed] [Google Scholar]
10. Smith BW, Chang KWC, Yang LJS, Spires MC. Comparative accuracies of electrodiagnostic and imaging studies in neonatal brachial plexus palsy. *J Neurosurg Pediatr* 2018;23 (1):119–24. [DOI] [PubMed] [Google Scholar]
11. Clarke HM, Curtis CG. An approach to obstetrical brachial plexus injuries. *Hand Clin* 1995;11 (4):563–80; discussion 580-581. [PubMed] [Google Scholar]
12. Michelow BJ, Clarke HM, Curtis CG, Zuker RM, Seifu Y, Andrews DF. The natural history of obstetrical brachial plexus palsy. *Plast Reconstr Surg* 1994;93 (4):675–80; discussion 681. [PubMed] [Google Scholar]
13. Ryan CS, Conlee EM, Sharma R, Sorenson EJ, Boon AJ, Laughlin RS. Nerve conduction normal values for electrodiagnosis in pediatric patients. *Muscle Nerve* 2019;60 (2):155–60. [DOI] [PubMed] [Google Scholar]
14. Antonovich D, Dua A. Electrodiagnostic Evaluation Of Brachial Plexopathies [Internet]. In: StatPearls. Treasure Island: (FL): StatPearls Publishing; 2021. [cited 2021 May 25]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK562197/> [PubMed] [Google Scholar]
15. Pondaag W, Malessy MJA. Outcome assessment for Brachial Plexus birth injury. Results from the iPluto worldwide consensus survey. *J Orthop Res* 2018;36 (9):2533–41. [DOI] [PMC free article] [PubMed] [Google Scholar]
16. Pondaag W, Malessy MJA. Evidence that nerve surgery improves functional outcome for obstetric brachial plexus injury. *J Hand Surg Eur Vol* 2021;46 (3):229–36. [DOI] [PMC free article] [PubMed] [Google Scholar]
17. Gilbert A, Pivato G. Obstetrical Palsy: The French Contribution. *Seminars in Plastic Surgery* 2005;19 (01):5–16. [Google Scholar]
18. Brochard S, Alter K, Damiano D. Shoulder strength profiles in children with and without brachial PLEXUS PALSY. *Muscle Nerve* 2014;50 (1):60–6. [DOI] [PMC free article] [PubMed] [Google Scholar]
19. Lagerkvist A-L, Johansson U, Johansson A, Bager B, Uvebrant P. Obstetric brachial plexus palsy: a prospective, population-based study of incidence, recovery, and residual impairment at 18 months of age. *Dev Med Child Neurol* 2010;52 (6):529–34. [DOI] [PubMed] [Google Scholar]
20. Kawabata H, Masada K, Tsuyuguchi Y, Kawai H, Ono K, Tada R. Early microsurgical reconstruction in birth palsy. *Clin Orthop Relat Res* 1987; (215):233–42. [PubMed] [Google Scholar]